Production of Heart Sounds by the Cardiac Catheter

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Extra heart sounds are frequently produced by the cardiac catheter in the human subject during routine cardiac catheterization. They are associated with a characteristic pressure artefact, which suggests that the sound is produced by back-and-forth movement of the catheter tip across the pulmonic valve. Similar sounds, and also murmurs, can be produced by catheter passage across pulmonary or aortic valves in excised canine hearts. The fact that such artificial sounds can be produced by the intracardiac catheter suggests that some caution must be used in interpreting phonocardiograms obtained during cardiac catheterization.

Increased interest in the heart sounds in recent years has led to attempts to obtain better correlation between the sounds and mechanical events within the heart than can be provided by external recording of pulses and sounds. One method is the simultaneous recording of external heart sounds and intracardiac pressures during cardiac catheterization. Recently the recording of intracardiac sounds by means of the intracardiac phonocatheter has been used in an attempt further to refine interpretation.1,2 Such methods have proved to be very useful.

For the past year we have been recording simultaneously external heart sounds and intracardiac pressures in a large proportion of routinely catheterized patients. We have observed that heart sounds may be produced by movement of the cardiac catheter within the heart, and that such sounds may lead to errors in interpretation of the phonocardiogram obtained during catheterization. Because of the increasing use of phonocardiography during cardiac catheterization, especially with the intracardiac phonocatheter, we think it of some value to report our findings regarding the incidence, characteristics, and mechanism of production of such catheter-induced heart sounds.

Material and Methods

Of a total number of 132 patients catheterized in this laboratory over a period of a year, 57 will be considered. All these subjects had simultaneous recording of intracardiac pressures and heart sounds during withdrawal of the cardiac catheter from the pulmonary artery to right ventricle and atrium.

Intracardiac pressure pulses were recorded photographically by means of a multichannel mirror oscillograph via the catheter and a Statham strain-gage transducer (P23D). The heart sounds were received by a microphone (Cambridge Instrument Co.) placed on the anterior chest wall in the pulmonic area or along the left sternal border, amplified by means of a low-level amplifier (Tektronics, Inc., Type 122), and recorded by the same oscillographic recorder. With this equipment pressure events on the recording have a delay of no more than 0.01 second in relation to the phonocardiographic events, even with the smallest and largest catheters used.

Clinical Observations

The records of 18 of the 57 patients studied were found to show an extra heart sound. In all cases, this was loudest along the left sternal margin at the second or third intercostal spaces. The sound was clearly audible to auscultation over this area.

In 17 of the 18 cases, the extra sound had the following characteristics: 1. It had the same pitch and duration as a second heart sound. 2. It was noted only when the catheter tip appeared to be in the right ventricular outflow tract, or during withdrawal of the catheter from pulmonary artery to right ventricle. In 7 cases, the extra sound was noted only in 1 or 2 heart cycles during the withdrawal, but in the other 10 cases it occurred repeatedly during the period in which the catheter tip seemed to be in the right ventricle. 3. It occurred in early diastole as shown in the example in figure 1. There was no fixed
The extra heart sound (x) in diastole is induced by the cardiac catheter in a human subject. The pressure tracing represents a combination of pulmonary artery pressures (in later systole and in early diastole before the sound) and right ventricular pressures (in diastole following the extra sound and in the first portion of systole). The sharp drop in pressure coincident with the extra sound is due to the sudden pulling back of the catheter tip into the right ventricle.

We have interpreted these findings as follows. During end-diastole the catheter tip is in the right ventricular outflow tract, ventricular diastolic pressure being recorded. Systolic contraction of the ventricle pushes the catheter tip through the opened pulmonary valve, so that pressure in the pulmonary artery is recorded. After the second heart sound, in early diastole, the catheter tip remains in the pulmonary artery for a time; but then, as the ventricular cavity enlarges during diastole, it is pulled back through the now closed pulmonary valve into the ventricle, so...
that the recorded pressure drops suddenly. The sound is related to the same events. The presence of the catheter does not prevent a normal second pulmonic heart sound, but withdrawal of the catheter produces an extra valvular sound.

We have on several occasions noted such a back-and-forth movement of the catheter tip across the pulmonary valve during high-speed cineangioangiographic studies.

Other factors that might have affected the production or incidence of this extra sound were considered. The sound occurred with all sizes (5F to 10F) of cardiac catheters. Age seemed to be no factor, since the range was from 4 to 47 years. The extra sound occurred with a variety of cardiac abnormalities, including 6 with atrial septal defect, 5 with interventricular septal defect, 4 with mitral stenosis, and 2 with primary pulmonary hypertension. There seemed to be no relationship to left-to-right intracardiac shunts, since 6 patients had no shunts, 7 had moderate-to-large shunts, and 4 had balanced left-to-right and right-to-left shunts. The 1 factor that appeared to be of some importance was that of pulmonary hypertension, a pulmonary artery systolic pressure of greater than 40 mm. Hg being present in 12 of the 17 cases, a greater incidence than would be expected from the material usually studied in this laboratory. Nevertheless, the existence of pulmonary hypertension was not essential for the produc-

![Fig. 3. An extra sound is absent when the catheter tip is fully in the pulmonary artery (first 2 cycles), but it appears when catheter tip passes back and forth between pulmonary artery and right ventricle.](image)

![Fig. 4. Sounds and "right ventricular" pressure tracings from 3 human subjects showing appearance of extra heart sound (x) and pressure artifact coincident with sound.](image)
EXPERIMENTAL OBSERVATIONS

In view of the findings in patients, we thought it desirable to attempt the experimental production of heart sounds by means of the cardiac catheter, and to verify the valvular origin of the sound.

1. The intact hearts and great vessels from 2 recently killed normal dogs were used. In each, the pulmonary artery was cannulated and attached by means of plastic tubing to a small reservoir of water. A pressure of approximately 25 to 30 mm. Hg was maintained in the pulmonary artery, which kept the pulmonary valve cusps tightly closed.

Pressures within the right ventricle and pulmonary artery were recorded by means of a cardiac catheter inserted through the right ventricular wall. Sounds were recorded by means of the equipment described above, the microphone being carefully placed over the main pulmonary artery. During the procedure, pressures and sounds were recorded simultaneously while the catheter tip was being moved slowly or rapidly in and out of the pulmonary artery, through the pulmonic valve.

In both hearts the passage of the catheter tip through the pulmonary valve, in either direction, caused loud sounds to occur. An example is shown in figure 6. The high intensity of sound at the pick-up site is demonstrated by comparison with the apex sounds of a normal human adult taken with the same equipment and at the same amplifier setting.

The main factor in the production of the sound seemed to be the setting of the tensed valve cusps into vibration by insertion or withdrawal of the catheter tip. The size of the catheter was unimportant as far as sound production was concerned, as was the position (lateral or central) of the catheter in the valve. The speed of withdrawal was important: when fast, a sharp sound was generated; when slow, no sound was recorded but the presence of the catheter tip in the valve produced pulmonic insufficiency and the associated murmur (fig. 7).

2. A similar experiment was performed with another excised canine heart with use of the aorta valve. Here again, passage of the catheter tip across the valve yielded sharp, loud sounds.

DISCUSSION

It is apparent from these findings that the presence of a catheter in the heart can bring about the production of true valvular heart
CATHETER PRODUCED HEART SOUNDS

Fig. 6. Sounds generated at the pulmonic valve in an excised dog heart when catheter tip passed rapidly back and forth between right ventricle and pulmonary artery. Pressure tracing shows abrupt fall in pressure as catheter tip passes across closed pulmonic valve. Lower tracing of normal adult human apex sounds taken with same equipment and amplification shows the high intensity of the sounds induced in the dog heart.

sounds. The coincidence of the pressure artefact and the extra heart sound, and the experimental findings are strong evidence in favor of the postulated mechanism as the cause of the diastolic extra sound found in most of our subjects. Further support comes from the demonstration by high-speed cineangiocardiography of a back-and-forth movement of the catheter tip across the pulmonary valve.

Since sounds were caused in the excised hearts both by insertion and by withdrawal of the catheter across the pulmonary valve, it may be questioned why only withdrawal caused a sound in the human subjects. The explanation undoubtedly lies in the fact that in the excised heart the valve was closed during both insertion and withdrawal, and was therefore capable of producing sound with both, whereas in the beating heart, systolic opening of the valve occurred before ventricular contraction pushed the catheter through, and therefore no sound could be generated.

It is interesting that no diastolic sounds were produced in cases with pulmonic valvular stenosis. We have no definite explanation for this observation, but there are 3 possibilities: 1. The pressure in the pulmonary artery is insufficient to tense the valve cusps enough to produce sound. 2. The rigid valve cusps are not able to vibrate sufficiently to cause audible sound. 3. Because of the narrow valve orifice the catheter tip cannot easily move into and out of the pulmonary artery, and the conditions for the production of the sound are not established. Cineangiocardiographic studies on suitable patients might help to settle this point.

The experimental findings, and the 1 patient with pulmonic stenosis who had a systolic sound, suggest that other sounds and murmurs can be produced by a catheter in the heart. No murmurs attributable to the catheter were recorded externally in any of our human subjects, but it may be that any murmurs produced were of insufficient intensity. The experimental findings demonstrated the striking loudness of the diastolic sound, which was easily recorded in patients.

In view of the increasing use of phonocardiographic technics during cardiac catheterization, one should be aware of the possibility of catheter-induced extra sounds, for under some circumstances the diastolic sound described could be interpreted as a late pulmonic second sound. Although the presence of the pressure artifact, which to our knowledge has not been described before, is helpful in making the proper distinction, many of the phonocatheters in use at the present time do not have a separate lumen for measurement of intracatheter pressures, making this check on the sound events unavailable.
Lewis and co-workers, who have reported on the use of the phonocatheter in man, did not discuss the development of the extra sounds reported here, but did mention that "when the catheter tip was in contact with the inner wall of the heart or valves, loud knocking sounds were obtained."

The fact that such artificial sounds as those described can be produced by the intracardiac catheter suggests that some caution must be used in interpreting the findings of intracardiac phonocardiography in other respects. It is possible, for example, that systolic murmurs recorded in the pulmonary artery could be produced or accentuated by the catheter. In 1 patient, studied in this laboratory, with a stenosis of the right branch of the pulmonary artery, the presence of a catheter in the area of stenosis led to an accentuation of the murmur.

The frequency of extra sounds noted in this study indicates that the back-and-forth movement of the catheter tip across the pulmonary valve is not an unusual occurrence during cardiac catheterization, and, in fact, an additional 5 patients of the 57 studied showed the pressure artifact without recordable sound production. This catheter movement is of some practical importance in routine cardiac catheterization, for what is thought to be a high right ventricular blood sample may in reality be a mixture of ventricular and pulmonary artery bloods. Such a finding in a patient with patent ductus arteriosus might lead one to suspect wrongly a ventricular septal defect or pulmonic insufficiency.

**Summary**

In 18 of 57 patients who had phonocardiograms recorded during diagnostic cardiac catheterization, an extra heart sound was recorded. In 17 of the 18, the extra sound was in diastole, and could be identified by its timing and its association with a characteristic pressure tracing. Movement of the catheter tip back and forth across the pulmonic valve during the heart cycle is thought to be responsible for production of the sound and the pressure artifact. Experimental studies in excised canine hearts showed that catheter passage across the pulmonary or aortic valves induced similar sounds, and also murmurs.

In an eighteenth patient, one with valvular pulmonic stenosis, catheter movement in the pulmonary artery appeared to be the cause of an extra heart sound in systole. The diastolic sound was heard in none of the 10 patients with pulmonic stenosis.

The fact that such artificial sounds can be produced by the intracardiac catheter suggests that some caution must be used in interpreting phonocardiograms obtained during cardiac catheterization.

The frequency of the back-and-forth movement of the catheter across the pulmonary valve suggests that not uncommonly what is thought to be a high right ventricular blood sample is in reality a mixture of ventricular and pulmonary artery bloods. This would be of some importance in the cardiac catheterization of patients with patent ductus arteriosus.

**Summary in Interlingua**

In 18 ex 57 patientes, in qui phonocardiogrammas esseva registrate durante diagnostic catheterismo cardiac, un supernumerari sono cardiac esseva registrate. In 17 del 18 casos, ille sono esseva in diastole e poteva esser identificate per le tempore de su occurrentia e per su association con un characteristic curva de tension. Movimentos del puncta del catheter in avante e in retro a transverso le valvula pulmonic durante le cyclum cardiac es conside-rate como responsabile pro le production del sono e le artefacto de tension. Studios experimentala in excidite cordes canin monstrava que le passage de un catheter a transverso le valvula pulmonar o le valvula aortica induceva simile sonos e etiam murmures.

In le dece-octave patiente, qui habeva stenosis del valvula pulmonic, le movimento del catheter in le arteria pulmonar pateva esser le causa de un supernumerari sono cardiac in systole. Le supra-discutite sono diastolic esseva audite in nulle de 10 patientes con stenosis pulmonic.
CATHETER PRODUCED HEART SOUNDS

Le constatazione che tal sonos artificial pote esser producete per le catheter intracardiaci suggere que certe precautiones debe esser usate in interpretar phonocardiogrammas obtenite durante catheterismo cardiac.

Le frequentia del movimento in ante e retro que le catheter executa a transverso le valvula pulmonar suggere que il occurre non infrequentemente que un specimen de sanguine reguardate como de origine dextero-ventricular alte es in realitate un mixtura de sanguine ventricular con sanguine de arteria pulmonar. Isto esserea de aliquun importantia in le application de catheterismo cardiac al studio patientes con patente ducto arteriose.

REFERENCES

Incidence of the Disease.—As noted long ago by Sir Gilbert Blaine, angina pectoris is a rare affection in hospital practice. Gairdner criticises this statement rather sharply, and yet I think that a majority of hospital physicians would be found to support it. During the ten years in which I lived in Montreal, I did not see a case of the disease either in private practice or at the Montreal General Hospital. At Blockley (Philadelphia Hospital), too, it was an exceedingly rare affection. I do not remember to have had a case under my personal care. There were two cases in my service at the University hospital. During the seven years in which the Johns Hopkins Hospital has been opened, with an unusually large “material” in diseases of the heart and arteries, and with many cases of heart pain of various sorts, there have been only four instances of angina pectoris. You will find the statement in Fagge’s Practice (third edition, vol. ii, p. 26) that the “writer has never seen classical angina in hospital practice.”

On the other hand, an individual consultant may see within a year more cases than occur in all the hospitals of his town within the same period. In corroborration of this striking contrast between the incidence of angina pectoris in hospital and consulting work I may refer to the statistics of the Edinburgh Royal Infirmary, in which for the two years covered by the Hospital Reports, 1893 and 1894, there were five cases among a total of 8,868 medical cases. Compare with this the personal experience of the distinguished Edinburgh consultant, Dr. Balfour, who, in his recently issued work on the Senile Heart, gives an analysis of ninety-eight cases of angina pectoris seen within ten years. My individual experience embraces a series of sixty cases, forty of which may be regarded as true angina.—WILLIAM OSLER. Lectures on Angina Pectoris and Allied States, 1897.
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